HYSTERICAL STIGMATA CAUSED BY ORGANIC BRAIN LESIONS.*

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In introducing for your consideration the subject of the origin of hysterical stigmata in organic brain diseases I am fully aware of the fact that I offer nothing new, but I am actuated by the desire of helping along the movement of trying to trace all of the so-called functional diseases to an organic basis.

In treating of the nature of hysteria and commenting upon the fact that it is a psychosis, the majority of writers are carried away by the idea that the mind is something wholely apart from the brain, and that mental aberrations are wholly independent of physical changes. As our knowledge increases we find more and more that functional use is attended with physical changes; even abstract mental activity is accompanied by increased metabolism, as is proven by an increase in the consumption of oxygen, and an increased exhalation of carbonic acid gas. We know that the child is born with all the faculties of the soul latent; that as the brain develops and as the general and special senses carry impressions to it the soul gradually develops the faculty of intellect and will, and in old age these faculties deteriorate when the brain itself undergoes retrograde metamorphosis. I need refer only to progressive paralysis of the insane to show how the soul or mind is deprived, one after the other, of its faculties until finally the individual is reduced to a vegetative existence.

That there is an interdependence of mind and body is perfectly apparent, but the manner or kind of relationship is still beyond our knowledge, most authorities assuming the doctrine of a psycho-physical parallel: that psychic phenomena go hand in hand with certain physical conditions, and that the perfection of the former is dependent upon the physiological and anatomical integrity of the latter.

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Before the seventeenth century the wandering uterus played a great rôle in the etiology of hysterical symptoms. The French in the seventeenth century first looked upon the brain as the seat of hysteria. Up to the middle of the nineteenth century hysteria was looked upon as a reflex neurosis. To-day the opinion is universal that hysteria is a general neurosis. Charcot, in developing his doctrine of the relation of hysteria and hypnotism made clear the relation of some of the symptoms to certain psychic changes. But Schultze emphasizes the fact that the psychology of hysteria demands much further elucidation and investigation.

If hysteria is a psychosis, then we must admit that while many of its manifestations are due to localized disturbances of cortical functions, the cortex of the entire brain is affected in its manifestations.

Psychic activity which represents the highest and most complicated manifestations of life demands of the neurone not only the highest development of structure, but also of function.

We are unable to-day, in a measure at least, to solve the physiological problem of changes in the neurone on which psychical manifestations are based. We know that the neurone reacts to external stimuli in two ways which are opposed to each other.

In the first place, stimulation of a neurone produces *external* manifestations of neuronic activity, which show themselves in muscular contraction, heat production, secretion, etc.

In the second place, stimulation affects the neurone in such a way as to limit or curtail the energy produced.

These effects are respectively known as the conducting and the inhibitory activity. Both of these activities are dependent upon definite bio-chemical and bio-mechanical activity.

Verworn holds that the protoplasm of the ganglionic cell consists of numerous living molecules of albumin, which are constantly performing either positive or negative molecular work, and sometimes one and sometimes the other predominates. The positive molecular activity manifests itself in an oxidation process, viz., the using up of the protoplasmic molecules in the production of the specific impulse of the cell. The negative molecular action is one of assimilation, a synthetic process, namely, the building up of the complex chemical molecules of the neurone.

Fatigue and exhaustion, we shall see, result from an excessive positive molecular activity, recuperation from an excess of negative molecular activity. Usually a neurone is at the same time under the influence of both stimuli. Its activity shows itself either in the nature of urging on or increasing an impulse which is already generated, or in diminishing the force of such an impulse.

The collision of two impulses in the same neurone is called "the interference of the stimuli." The circumstances which determine the result of such an interference, whether there shall be an increase or a diminution of activity, depends upon the condition of the neurone.

In a normal condition of the neurone the result of such an interference is usually inhibition; in an exhausted neurone there is usually an increase of irritability or activity.

The manifestations of a normal neuronic activity are always, therefore, somewhat inhibited.

Abnormal neuronic activity may either show itself in the form of an explosion or its activity may be entirely absent, due either to a complete exhaustion caused by a want of working capacity or it may result from complete inhibition due to the excessive manifestation of the "interference of stimuli."

Wundt says that in every central neurone we must distinguish physiologically between a central zone and a peripheral zone of the cell.

The central zone (nucleus) is the seat of negative molecular activity, viz.: anabolism and the peripheral zone is the seat of positive molecular activity, viz., katabolism. Any stimulus reaching the central zone increases the negative molecular activity, which is inhibitory in character, and causes this activity to overflow over the peripheral zone; and *vice versa,* any stimulus acting upon the periphery of the zone increases positive molecular activity, which produces increased activity and causes it to spread over the central zone. The central zone, according to Wundt, corresponds to the origin of the axis cylinder fibrils, whereas, the peripheral zone corresponds to the origin of the protoplasmic masses of the dendrons. I will not go into the theories and experiments calculated to study and demonstrate the generation of impulses in the single neurone, but will call your attention to careful experimental work, showing the effect of functional activity and fatigue upon the structure of the ganglion cell. In this respect the work of Guerrini stands out most prominently.

Guerrini took twelve dogs and placed them in an apparatus similar to a squirrel's cage and made them travel distances varying from 21 to 60 miles. Some of these animals were killed at once and others, used as control animals, were fed and allowed to rest before being killed. Both groups of animals were examined in precisely the same manner, same methods, stains, etc. The control animals were found to be perfectly normal. In the fatigued animals organic changes were found in the gray matter of cortex and cord, but especially marked in the psycho-motor area. These changes were as follows:

1. Enlargement of the peri-cellular lymph space, due to a diminution in the size of the cell body. 2. Accumulation of leucocytes in the peri-cellular lymph spaces. 3. Changes in the achromatic network of the cell body. 4. A dissolution of the chromophile masses. 5. Vacuoles in the protoplasm. 6. Irregularity in the contour, and a diminution in the size of the nucleus. 7. Vacuoles in the nucleus.

Hodge made similar experiments on the spinal cord of cats. He stimulated the motor root with an electric current on one side, and therefore had the opposite anterior horn always present for the purpose of control. He not only found all the above changes of Guerrini, but proved that these changes were directly proportioned to the length of the period of stimulation of the nerve-trunk; and that after ten hours of stimulation the nucleus was diminished in size 44 per cent. Hodge also examined the nervous systems of birds, bees, sparrows, swallows and pigeons in the morning after a night's rest, and at night after a day's work, and found the above differences between normal cells and cells showing fatigue.

The above changes in the ganglionic cells have also been studied in the living cell. The sympathetic nerve of a frog fed with a nutrient plasma was put under a microscope, stimulated with electricity, and the above cell changes were noted as a result of this stimulation.

It has also been shown that the above fatigue changes occur early and in a more profound manner if the nutrient plasma is not normal, but is changed by inorganic mineral poisons, the poisons of cymotic diseases, perhaps alcohol and urea, which it is needless to say is borne out by our every-day experience in practice. It has been found that if these cellular changes went beyond a certain degree it was impossible for the animal to recover completely. That excessive fatigue was accompanied by permanent changes in the structure of the cell.

Now what practical bearing have these researches upon the organic basis of hysteria.

Hysteria, notwithstanding the fact that it has been known and described from the dawn of medical times, is a disease whose real character is in many respects even now wrapt in obscurity. This is primarily due to the fact that hysteria manifests itself in the realms of the mind, not in such a manner as to show marked aberrations of the intellect, but in anomalies of character and emotions. Its innermost nature is masked by boundless and an unending variety of bodily symptoms of illness.

Its signs and symptoms are found to be interlaced with, exaggerating or masking, the signs and symptoms of all other diseases.

The primary fact which confronts us in contemplating the nature of hysteria is that it must be based upon a defective organization, upon molecular changes in the protoplasm of the gray matter of the cortex.

Charcot and his school represent the extreme view that the primordial cause, compared to which all other causes are exciting causes only, is heredity. Hysteria is a distinct family disease, handed down usually from mother to children, although it may descend through the father. In the ascendancy of the hysterical subject we can, as a rule, trace nervous affections which need not always be hysteria, but some other functional neurosis.

While this opinion of Charcot is almost universally held to-day for ordinary hysteria, it does not hold for traumatic hysteria. Oppenheim, than whom there is no better authority on this special subject, is of opinion, and every man who has seen many cases will agree with him, that the majority of cases following accidents occur in individuals who have previously been entirely healthy, and in whose family history no trace of nervous diseases can be found. Therefore, in these individuals some change in the gray matter of the cortex which has produced this altered condition of the emotions, as well as the hysterical stigmata, must have been acquired as a result of the accident.

Hence, we can conclude that whatever may be the underlying change in the cortex of the brain in hysteria, it may either be inherited or acquired.

Charcot and the French school incline more to the psychic nature of hysteria, comparing hysteria to the artificial state produced by hypnotism.

Solier has lately advanced the theory that hysteria is a state of somnambulism which, instead of enduring for a few minutes or hours, continues in hysterical persons for days, weeks and even months (Vigilambulism). These conditions of lethargy may affect the brain as a whole, or they may affect isolated motor, sensory or visceral areas of the cortex. In proportion as there is a slight or deep, a general or localized degree of somnambulism, we have slight or marked localized or general manifestations of hysteria. This theory of somnambulism or sleep presupposes that state of the cortex which leads to sleep, and we can easily imagine a state of the ganglion cells similar to that found by Guerrini in dogs, and by Hodge in birds, cats and insects.

In hysteria we have a pathological state of the nerve centers whose chief clinical manifestation is an incoördination of brain functions, where the emotions influence the will power more strongly than is normally the case.

Oppenheim's explanation of the nature of hysteria brings us closer to our anatomical ideas of the disease. He says that "the nature and origin from which all hysterical phenomena spring is an irritable weakness of the nervous system, an abnormal irritability, and an abnormal exhaustibility of the gray cortical centers." This affects all the functions of the nervous system, but shows itself first and most actively in the sphere of the emotions.

Both external and internal stimuli produce greater emotional effects in the hysterical than in the healthy. The manifestations of hysteria upon the motor, vaso-motor, secretory and sensorial functions are merely an exaggeration of what occur under ordinary physiological conditions. The effect which the emotions usually have upon these various functions is modified by education, practice and will power. That the emotions do affect these various functions, and that there is a close relation between them, is seen, for instance, in the mimical contractions of the facial muscles during laughter or crying, the shedding of tears, the blush of shame, the pallor of anger, the tonic muscular contraction of anger, the trembling of fear.

The effect of the emotions upon the various functions depends partly upon the stimulus producing the emotion and partly upon the state of irritability of these various centers. The response is pathological when the effect is out of proportion to the cause, and the fundamental cause of an excessive effect is an irritable weakness of, and a consequent abnormal ease, with which the motor, vaso-motor, sensory and secretory functions are brought into action.

The pathological phenomena of hysteria are merely an expression of increase in the physiological action of these centers. The laughing and crying fits of the hysterical correspond to the laughing and crying of the healthy; the general convulsions find an analogy in the trembling of fear, in the tonic muscular contractions of anger. Irritations which the normal individual does not perceive at all, or which at most produce a sense of discomfort, produce in the hysterical pain, paresthesia, globus and increased rapidity of the heart's action.

All signs and symptoms can be explained by an irritable weakness, an abnormal exhaustibility of function of the brain cortex. I have given you Oppenheim's views as to the nature and origin of hysterical phenomena rather extensively because they coincide with my views on the subject, and because, if they are true, they take us one step nearer to a solution of the question upon a basis of the results obtained in animal experiments.

If irritability and abnormal exhaustibility, a lack of mental and emotional inhibition are characteristic of hysteria, we may assume that the same protoplasmic changes of the ganglionic cells which we assume to be present in neurasthenia are also present in hysteria, for we know that hysterical manifestations are in inverse proportion to the health of the individual. The better the general health the less manifest the symptoms. The greater the exhaustion, the greater and more certain the effects of suggestion, even in hysteric experiments. The greater the exhaustion the more numerous and more tenacious the symptoms and signs.

Let me introduce at this point two cases:

Case No. 1.—Mrs. A., light mulatto, has never borne children. Has been nervous since 1893, when she was injured in a railroad accident. Recovered from this accident completely after six months.

Denies syphilitic infection, although she bears evidence of syphilis in nodes and pigmented scars.

Has never had hysterical convulsions or hysterical stigmata.

On Dec. 12, 1902, while sitting and talking she was seized with a feeling of numbness in the right hand and arm, fell from her chair to the floor and was unconscious for a few minutes; when she recovered consciousness she found that she was unable to speak, and that her right side was paralyzed. On the next day she was able to speak a little, and there was some slight movement in the limbs. Was brought to the neurological department of the Cincinnati Hospital on the third day.

On admission, patient gave the above history; was not aware of the existence of any sensory disturbance further than that the side was numb.

Examination.—Female, middle age, well preserved; some copper-colored spots about the size of a silver dollar over both tibia.

Intelligence good, no speech defect, somewhat emotional, and desirous of much attention and sympathy.

Slight ptosis of left eyelid, left pupil smaller than the right. Pupils normal in reaction to light and accommodation.

No defect in the external muscles of the eyes.

Field of vision not tested.

Right labio-nasal fold absent, right angle of mouth lower than left; on opening mouth right angle of mouth depressed, left drawn upward. Tongue protruded toward the right.

No spastic condition of tongue or face.

Almost total paralysis of right arm.

Right leg can be moved, but is very weak.

Reflexes, both radial and patellar, are much diminished on right side, normal on the left side.

There is complete analgesia of the right side of the body, going sharply to the median line of the scalp, the middle of nose, chin, sternum and abdomen in front and on back down along the spinal processes. This analgesia affects the entire right side of the body. The sense of touch is present. There is also a complete loss of temperature sense. Stereognostic sense lost in the right hand. Muscle sense defective in fingers and toes, but normal for limbs.

Sense of smell and taste are normal.

During the next ten days there was a gradual improvement in the hemiplegia, but the sensory disturbance remained unchanged. Daily attempts to remove the hemianesthesia by mere verbal suggestion were ineffectual. After two weeks of failure by verbal suggestion a strong Faradic current was used. A promise was made that the electricity would put new life in the affected side. Vigorous contractions were produced in the region supplied by the facial nerve and brachial plexus, and was followed by quick return of sensation in the right side of head and arm, both for pain and temperature. The current was not applied to leg, and although the sensation improved, it was not until the next day, when the leg was also treated, that the sensation returned to a normal condition.

In the presence of the class the hemianesthesia was reproduced on two occasions by suggestion, and made to disappear again by the use of the Faradic current.

On the tenth day Babinski sign was marked in right foot, absent in left. Became more marked after return of sensation.

One month after onset of hemiplegia well-marked ankle clonus of right foot; patient is still able to walk; tongue is protruded slightly to the right, paresis of right side of face almost completely gone. Marked weakness of right leg. To sum up in a few words:

Onset, with apoplexy and short loss of consciousness of a right-sided hemiplegia, tongue, arm and leg, gradually decreasing with development of increased reflexes; Babinski sign and ankle clonus in the fourth week.

Complete hemianesthesia for pain, temperature and partly of muscle sense, which remained unchanged for two weeks, and then was made to disappear completely and almost instantly by the combined verbal and electrical suggestion.

The patient is most positive in her statement that the disturbance of sensation did not exist before the attack of apoplexy, although she was sharply questioned about the injury to the left shoulder joint ten years before. She stoutly maintains that the sensation was perfectly normal.

Case N. 2.—Cerebro-spinal syphilis with hysteria. Henry Y., aet. 38, single, waiter. Entered Cincinnati Hospital Jan. 24, 1905; complaint, weakness of legs. Denies syphilis; has had ordinary diseases.

Trouble began four weeks before admission. Weakness of right leg, felt weak; he was compelled to walk in the way described below, and he could not change his gait as long as his mind was occupied with his right leg.

Had double vision; incontinence of urine when great necessity for urinating was present.

Examination.—Mental condition apprehensive, fear of paralysis.

Pupils unequal, do not respond to either light or accommodation. Ptosis of right eyelid, weakness of right side of mouth, no disturbance of sensation of face or tongue. Arms normal.

Weakness of both legs, especially the right. Increased reflexes, ankle clonus of right side, Babinski both sides.

Patient had a peculiar gait, which resembles the "two-step" dance, and is known as the "two-step man."

The organic symptoms rapidly diminished under the anti-syphilitic treatment. After three weeks the gait became normal, and has never had a recurrence of the dancing movements. To-day the pupils react normally and all organic signs have disappeared, with the exception of a slight spastic paresis of the right leg, with ankle-clonus, but notwithstanding this the "two-step gait" has never recurred.

The combination here of a hysterical gait superimposed upon a mild case of cerebro-spinal syphilis cannot be doubted. The interesting feature is that the entire condition was not looked upon as hysterical, a mistake easily avoided by making a careful examination.

It is needless to say that the diagnosis of hysteria alone would have been a calamity to the individual, who is well to-day and has returned to his usual occupation.

My object in introducing for your consideration the subject of the origin of the stigmata of hysteria from organic brain insults is to call your attention to a probable development of hysterical stigmata upon an organic basis.

We know that functional aberrations of ganglionic cells are due to some changes in the protoplasm of the ganglionic cells. For function of cells and chemical change are inseparable, combustion, oxidation of substance, using up of molecular parts, metamorphoses, are the physical basis of function; constructive and destructive metamorphoses are always going hand in hand; like in neurasthenia, we can assume that in hysteria there is a defective anabolism, either inherited or acquired, which leads to an imperfect organic state of the nerve cells. The ganglionic cell, either from its origin is structurally imperfect or it acquires a structural imperfection. Added to this condition is another which, while in itself is more or less an intangible condition, is dependent upon the protoplasmic perfection of the cell itself.

Each perfect cell has the inherent power of metabolism, of katabolism and anabolism, both of which are regulated by some unknown agency, which is perhaps inherent in the nature of protoplasm. In hysteria this function of metabolism is defective in two ways: in the first place, the reconstructive ability of the protoplasm of the cells is defective, either inherited or acquired, and in the second place, the protoplasm is irritably weak. There is a lack in the cell itself of normal inhibition. The function of the cell is either dormant or it does not act at all. There is an exhaustion of function and the protoplasm fails to produce energy, or we have the other manifestation; the energy produced, instead of being regular, rhythmic and sustained, occurs in the nature of an explosion. Both of these changes in cellular function, either a diminution or absence of function or an explosion of cell energy, are dependent upon an irritable weakness of the cell, which in turn depends upon a state of diminished nutrition, of structural imperfection. We know now, as a result of careful investigation into the physiology and pathology of nerve cells by numerous observers, that these organic changes of the nerve cell are brought about by ordinary daily work, by exhaustion and by various poisons and toxins, which are apt to be found influencing our bodies in every-day life, normally; and this has been proven experimentally, the cell protoplasm recovers from these organic changes as a result of taking food and rest. Thus in all the above experiments the changes noted in the cells were absent in control animals, birds, insects, after the normal period of rest.

But it has been proven by experiment that there is a certain point produced by exhaustion beyond which recovery does not take place, or only after a long time, and then imperfectly. These experiments would correspond to our clinical experiences, with neurasthenia, which we might term a chronic condition of exhaustion resulting from long-continued over-exertion, inadequate nutrition, inadequate rest, in which repair has not equalled consumption, and in which a lowered state of the organism as a whole has weakened, even under favorable conditions, the ability of the protoplasm to establish a normal metabolism and a normal balance of conservation and expenditure of energy.

In hysteria, I should imagine that this change in the organic structure and in the physiological balance of protoplasm is fundamentally the same as it is in neurasthenia, except that it is exaggerated. We see clinically in hysteria the same exhaustion, the same irritability of function; hysterical individuals are always neurasthenics, but they have neurasthenia to a highly exaggerated degree, with the added burden of inheritance. The pathological picture which I draw for myself of the organic basis underlying hysteria is that we simply have more profound protoplasmic changes in the cell, plus a greater degree of weakness of anabolism, leading to constant physiological irritability of function, manifesting itself either in exhaustion or explosion, or an excess or incoördination of physiological inhibition. This depends either upon an inherited or acquired defective condition of the protoplasm of the ganglionic cells of the cortex, not only of those presiding over the functions of motion, but also of general and special sensation or association, but especially of those ganglionic centers which preside over the vegetative and generative functions, of circulation, respiration, digestion, excretion, etc. This defective general state is either inherited or acquired, and the functions of the ganglionic cells are so enmeshed in the vicious circle of defective nutrition of protoplasm and excessive expenditure from functional incoördination that it is impossible for the system to establish a physiological and structural balance.

Rapidity of change, both psychical and physical is not necessarily opposed to the view of a molecular change in the ganglionic cells. These same characteristics are typically present in neurasthenia, and we can see only a difference in degree between the manifestations of neurasthenia and those of hysteria, only an involvement of a greater number of functions, a more widespread distribution of the condition in hysteria as compared with neurasthenia.

If, however, the stability of the stigmata, their fixed character and long duration are characteristic of hysteria, then we have all the more reason for assuming that they are dependent upon organic changes in the ganglionic cell similar in nature to those observed in animals after fatigue experiments.

And now I would like to enter upon the last point of this paper, namely, the production of hysterical stigmata by organic brain diseases.

It is needless for me to say that hysteria may enter into and mask the features of nearly all known diseases. It is especially apt, however, to associate itself with diseases which produce general exhaustion, or which are associated with the production of toxins, or which follow metallic or other chemical poisoning. I cannot digress, however, on account of the scope of this paper.

The organic diseases of the brain with which hysteria is usually found associated are those which produce general disturbances of circulation, increased intracranial pressure or general concussion.

The disease *par excellence* with which hysteria associates itself is multiple sclerosis. I have prepared for publication in another paper an exquisite case of this character, in which the hysterical element was so pronounced that notwithstanding the presence of nearly all the classical signs of multiple sclerosis, a mistake of diagnosis was made and the patient subjected to a course of treatment little short of cruel.

Hysteria is especially apt to be associated with brain tumor, and very often in the early stage of tumors located in latent brain areas it is not uncommon to make false diagnosis of hysteria. In cases of fracture of the base of the skull it occurs at times that we have hysterical stigmata superimposed upon the symptoms and signs due to organic brain lesions.

It is apparent, therefore, that hysteria associates itself with those conditions which alter the central nervous system, either by way of general exhaustion or intoxication or in which there is a general disturbance of the gray matter produced by inflammatory or circulatory changes. It is easy to picture to ourselves that multiple sclerosis, brain tumor, cerebro-spinal syphilitic meningitis, traumatic concussion, produce minute changes in distant cortical or sub-cortical centers, either by way of general pressure or circulatory disturbances. Pressure or disturbance of circulation produce a malnutrition of the ganglionic cell, which must resemble very closely the condition of the cells found in exhaustion by Guerrini, Hodge and others. That these changes in cells give rise to passing conditions of paralysis, anesthesia, etc., even under ordinary non-hysterical conditions we see daily in the transient signs and symptoms accompanying ordinary cases of organic hemiplegia. I need but call your attention in this direction to analogous fleeting conditions of hemiplegia, anesthesia, amaurosis, aphasia, which often appear in the uremic state of Bright's disease, and are commonly attributed to either localized edema or toxic conditions of the cortex, and which disappear almost as quickly and as spontaneously as similar hysterical conditions.

I assume, therefore, that hysterical signs and symptoms which we see accompanying organic diseases of the brain are the result of organic changes in the ganglionic cells, and that these organic cell changes result in a disturbance of their functions which lead clinically to that condition which we know as hysteria.

The principal end of these theoretical considerations is that hysteria should be treated, not as a pariah of diseases, but should have our careful attention, just as neurasthenia has. That while suggestion is useful, it is not curative.

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